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Volume IV AUGUST 1943 Number 8.

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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

PUBLISHED MONTHLY for the PHYSICIAN in the GENERAL PRACTICE of MEDICINE

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PHYSICIANS POSTGRADUATE PRESS
500 NORTH DEARBORN ST., CHICAGO, ILLINOIS

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

The Clinical Signs of Nerve Regeneration*

LEWIS J. POLLOCK, M.D.
Chicago, Illinois

The signs of regeneration of a nerve are the manifestations of recovery of function. Among these are return of sensation, both subjective and objective, disappearance of reaction of degeneration, increase of tone, disappearance of atrophy and return of motion.

These manifestations differ in appearance and rate of return as to the pathology of the nerve and as to whether recovery is spontaneous or is consequent to surgical intervention. They are dependent upon the condition of the neuraxones. If descending degeneration has been slight or absent and the nerve recovers spontaneously and rapidly, one type of course is followed; if resection and suture has been performed, another type is observed. If little or no degeneration has resulted, but a complete physiological interruption has existed for a long time, perhaps because of a constricting band, surgical relief of this morbidity is succeeded by a regeneration similar in character to that observed in lesions recovering spontaneously. If descending degeneration is severe or complete and conditions are such that the lesion recovers with no surgical interference, the course of recovery will be very similar to that observed following suture.

Many cases of complete physiological interruption of a nerve showed their first sign of regeneration at the time it would occur if the nerve were divided by the injury and sutured. From this point on the regeneration progressed exactly as it would in a sutured nerve. It is reasonable to assume that in this type of severe lesion, complete descending degeneration has occurred and conditions permitted the regeneration of the axones. Evidence of regeneration first appeared about the eighth or ninth month, and it was noticeable that a considerable number of men wounded at about the same time, all began to improve together.

In my opinion, this is additional evidence that great conservatism should be exercised in making a decision for resection and suture in severe lesions of peripheral nerves even though there was no recovery within 7 to 8 months.

The order in which the signs of regeneration appear have been given by Mme. Ath. Benisty[1] as follows: (1) Sensory regeneration, consisting of pain when the skin is pinched, pain when the nerve is pressed below the lesion, formication on pressure of the nerve and spontaneous aching in certain muscles; (2) arrest of atrophy and return of tonicity; (3) in some cases return of faradic contractility; (4) disappearance of objective sensory disturbances; and (5) voluntary movements.

In my experience pain upon pinching the skin was oftimes the first sign of nerve regeneration, but not in all instances. Only that return of pain to pinching which is found in

*From the Department of Nervous and Mental Diseases, Northwestern University Medical School.

such areas of skin as are outside the influence of nerve overlap, can be used as an indication of the recovery of a nerve. Frequently spontaneous aching and more frequently a sensation of a "different feeling" in an extremity preceded other signs of nerve regeneration. Pain upon pressure of the nerve trunk distal to the lesion was found unreliable.

Tinel's sign, or peripheral formication upon pressure or light percussion of the nerve trunk distal to the lesion, was found to be practically valueless. Where a nerve is superficial and pressure may be exerted upon it and it alone this sign might have some value. Unfortunately, only few of the peripheral nerves have a superficial course and this only for a short distance. Elsewhere other structures may be included in the pressure. Where the sign is elicited by light percussion, the concentric waves of motion transmitted from the percussed spot may stimulate the nerve at a considerable distance.

The visual observation of the degree of atrophy leads to faulty conclusion in all cases exclusive of the small hand muscles. In experimental animals it has been found that even measurements of the circumference of an extremity or displacement of volume of water does not correlate with the weights of denervated muscles. It is probably because of this inconsistency that I was unable from my material in the last war to use a lessening in degree of atrophy as a reliable sign of regeneration or to follow the progressive regeneration of a nerve by this method.

When tone was measured by a tonometer, it was found that only for a short time after injury to a peripheral nerve was the loss of tone any indication of the severity of the lesion. Even then the loss of the tone represented merely a reflection of the general loss of function. The difference in millimeters of mercury was expressed in a range of from 160 to 180 in normal muscle and 40 to 60 in the paralyzed ones. After a few weeks, infiltration, fibrosis, and other changes in the muscles and tendons vitiated what significance loss of tone might have.

Too little is known of the nature of trophic disturbances to enable us to employ them profitably in interpreting the severity of the lesion. Where protopathic sensibility was lost, trophic ulcers were likely to occur. When an extremity was immobilized, growth of nails ceased. When an extremity was protected by a dressing hypertrichosis was at times observed. Generalized atrophy of the bones indicated only disuse. In other words, the trophic disturbances can be employed as an indication of the severity of the nerve lesion only when judged in the light of the presence of other conditions.

There are three functions, the return of which give critical evidence of regeneration of a peripheral nerve. They are, the return of normal excitability of muscle and nerve to electrical stimuli, return of voluntary movement, and return of cutaneous sensibility.

The characteristics of the reaction of degeneration are well known and unnecessary to describe.

Regarding the electrical changes demonstrated by the methods personally employed, namely, faradic, and galvanic stimulation, it may be said that complete reaction of degeneration is always present in a severe lesion but does not indicate an irreparable one. Of all the changes to electrical stimulation, the slowness of the muscular contraction is the only constant phenomenon which can be satisfactorily employed in determining the reaction of degeneration. Polar changes are inconstant and of course it may be expected the response to faradism would be absent frequently, even in partial lesions. Especially is this true of cases requiring more than four months for recovery.

It has always been noted that during the early stage of degeneration of a muscle it is more irritable to galvanic stimuli. I believe this stage continues for a longer period of time than ordinarily described and investigation on this point merits attention.

When the amount of current necessary to produce a contraction progressively diminished after the initial period of "hyperexcitability," it indicates progressive recovery. When response to faradic stimulation occurs, recovery is almost complete.

An electric stimulus must reach a certain intensity before it will result in the contraction of a muscle. This minimal current, however, must be prolonged for a certain length of time to produce a response. At "infinite duration" there is a minimum strength below which no contractions occur (rheobasic voltage). As the duration is decreased the strength must be increased until a point is reached

where, no matter how strong the current, no contractions follow. The minimal duration of current necessary to produce a contraction with twice the rheobasic voltage has been designated empirically as the chronaxie of the tissue. The chronaxie of a human muscle with an intact nerve supply has been found to be 0.00016 second, whereas that of one whose muscle has degenerated is about 0.01 second. It was hoped that measurements of the chronaxie would furnish accurate information of diagnostic and prognostic value in peripheral nerve injuries. Although they have served to give precise measurement of the functional value of nerves and muscles in physiology, because of the inaccessibility of accurate instruments and the unreliability of certain others, few data are available which permit of critical judgment of its clinical value. When measured by the condenser system of Lewis Jones, it has failed to impress the American investigators with its reliability. More accurate methods, such as the Lucas pendulum, Lapicque's chronaximeter, Strohl's érgersimétre and Sachs' and Malone's chronomyometer have appeared to give accurate information in their several investigator's hands. It is possible that they may lead to profitable clinical investigations in the future.

Numerous other methods of electrical stimulation are being studied and it is hoped will give valuable indications of regeneration. Among these are progressive currents and radio frequency sine waves. The return of voluntary motion in a formerly paralyzed muscle is positive proof of regeneration of the nerve. The evidence of voluntary motion of a muscle is inferred from the movement of segments about the joint.

The frequency with which more than one muscle may produce a similar movement of the segments about a joint emphasizes the necessity for the use of great care in the analysis of all muscle movements. This care is the more necessary in the study of peripheral nerve lesions because the muscles under consideration may receive their nerve supply from different sources.

The preservation of certain movements, the loss of which is supposed to follow particular nerve lesions, has been observed for many years. These movements may be caused by a number of factors. Among these may be included the anastastomotic supply of muscles from adjacent nerves, movements produced by muscles other than primary movers in this action, movements occurring as the result of mechanical factors producing a change of direction of leverage by shortening and lengthening of tendons and muscles passing over several joints, and slight movements resulting from the recoil of elastic tissue following a movement in a direction opposite to the one desired. A few of the movements made possible by supplementary movements may be mentioned as illustrations: in radial nerve paralysis, extension at the wrist by flexing the fingers and contraction of the pronator radii teres (Fig. 1) in median nerve lesions, opposition of the thumb to the little finger by the adductor pollicis, opponens minimi digiti, and flexor brevis pollicis, in ulnar nerve palsy, adduction of the index finger by the extensor indicis. So great does this influence the movement of some segments that I have never been able to state definitely that such movements as I have observed return following resection and suture of the ulnar nerve, were unquestionably due to nerve regeneration, with the exception of a distinct contraction of the flexor carpi ulnaris. These movements are likewise very confusing in median nerve lesions. Some of the movements which cannot be supplemented

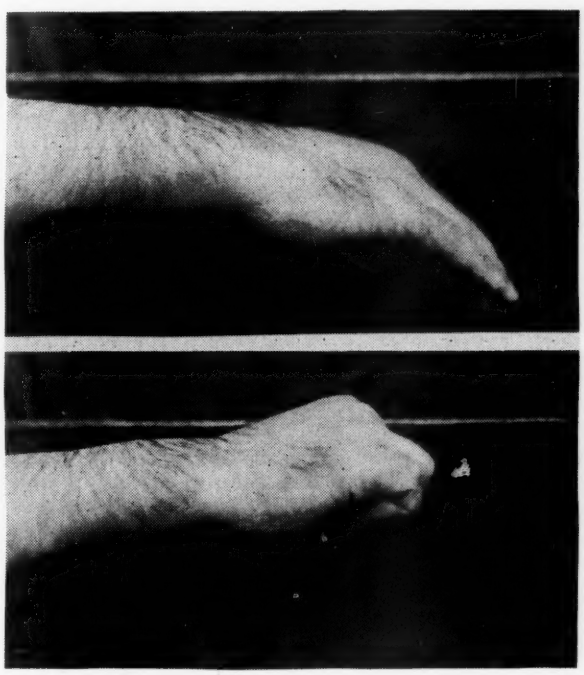

Figure 1. Extension at the wrist in radial nerve paralysis by supplementary movements produced by flexing the fingers.

in the various nerve lesions are: In radial nerve lesions, extension of the proximal phalanx of the thumb and abduction of the thumb in the plane of the palm, and extension (not alone tension) of the proximal phalanges of the fingers. In ulnar nerve lesions, flexion of the proximal phalanges of the ring and little fingers with the distal phalanges extended, and lateral movements of the extended middle finger: In median nerve lesions, flexion of the distal phalanx of the index finger and of the thumb. In combined lesions of the ulnar and median nerves, all movements of the hand except flexion at the wrist and hollowing of the hand. In external popliteal lesions, eversion of the foot.

The order in which movement is restored to muscles paralyzed as the result of peripheral nerve lesions and the particular muscles involved in partial or dissociated nerve lesions have a sufficient constancy to attribute to each nerve a clinical individuality. There is some difference of opinion among those who have studied this question as to this order of recovery, but my observations are in the respective nerve lesions as follows:

In ulnar nerve lesions, recovery was first seen in the flexor carpi ulnaris, then the small muscles of the hand. Because of supplementary motility, accurate measurement of the flexor profundus could not be made. Of the small muscles of the hand, the adductor of the thumb recovered more frequently than the abductor of the little finger.

In median nerve lesions recovery occurred first in the flexor carpi radialis and flexor longus pollicis. Movement seemed to return early in the flexor of the index finger, but from a study of residual paralysis, it was evident that flexion of the index finger remained imperfect for a long time, comparable to the opponens.

In radial nerve lesions the extensors of the wrist were first to recover, then the extensors of the thumb, then of the fingers. Extension of the middle finger returned before that of the index finger.

In sciatic nerve lesions the order of return of function was the gastrocnemius, tibialis posticus, peronei, tibialis anticus, extensor longus digitorium and then the flexors of the toes.

In external popliteal nerve injury the order of recovery was the tibialis anticus, peronei, extensor longus digitorum, extensor proprius hallucis.

For many years it has been noted that the total loss of sensation is limited to a much smaller area than one would expect from its anatomical distribution. It has been well recognized that the residual sensibility of a nerve is more extensive than its accepted anatomical distribution.

It has likewise been noted that after about 50 days following suture of a nerve, the area of analgesia begins to shrink. This return of sensibility to pain has been attributed to an early regeneration of protopathic fibers.

In some previous publications I stated my opinion that this early and dissociated return of sensibility to pain is due to the assumption of function of adjacent uninjured nerves. It is felt that this is true because: First, the return of sensibility to pain always occurred in certain areas of skin supplied by the various nerves. Second, when one or more adjacent nerves were injured simultaneously, sensibility to pain never returned in the borders between these nerves, where it usually returned when either of these nerves was injured alone. Third, when there was a return of sensibility to pain in a region of sensory distribution of a severed nerve this region became analgesic when an adjacent nerve was severed, for example, to be used as a cable transplant. Fourth, when sensibility to pain had returned in a region in the area of sensory distribution of a severed nerve, subsequent resection and suture of this nerve was not followed by a loss of this sensation. The return of sensibility to pain in such cases follows a pattern so characteristic that it may be recognized at sight. It never returns in the distal phalanges of the little finger in ulnar nerve lesions, never in the distal phalanges of the index and middle fingers in median nerve lesions, and so on. It always occurs along the borders of an uninjured nerve and may well be described as a shrinkage (Fig. 2).

Contrasted with this, the pattern in recovering nerves is strikingly different. Although shrinkage occurs, other changes are also present. The characteristic features of the sensory recovery of regenerating nerves may be enumerated as follows:

Return of sensibility to pain, touch, or temperature sense in that area of the sensory

distribution of a nerve which is supplied by it alone, i.e., the isolated sensory area of the nerve.

Return of sensibility to pain, touch, or temperature sense in patches some distance from the area supplied by an adjacent uninjured nerve.

Return of sensibility to pain, touch, or temperature sense in deep indentations.

Diminution of degree of loss of sensation of pain, touch, or temperature sense in the isolated sensory supply of a nerve.

Return of sensation of pain, touch, or temperature in the border between the sensory

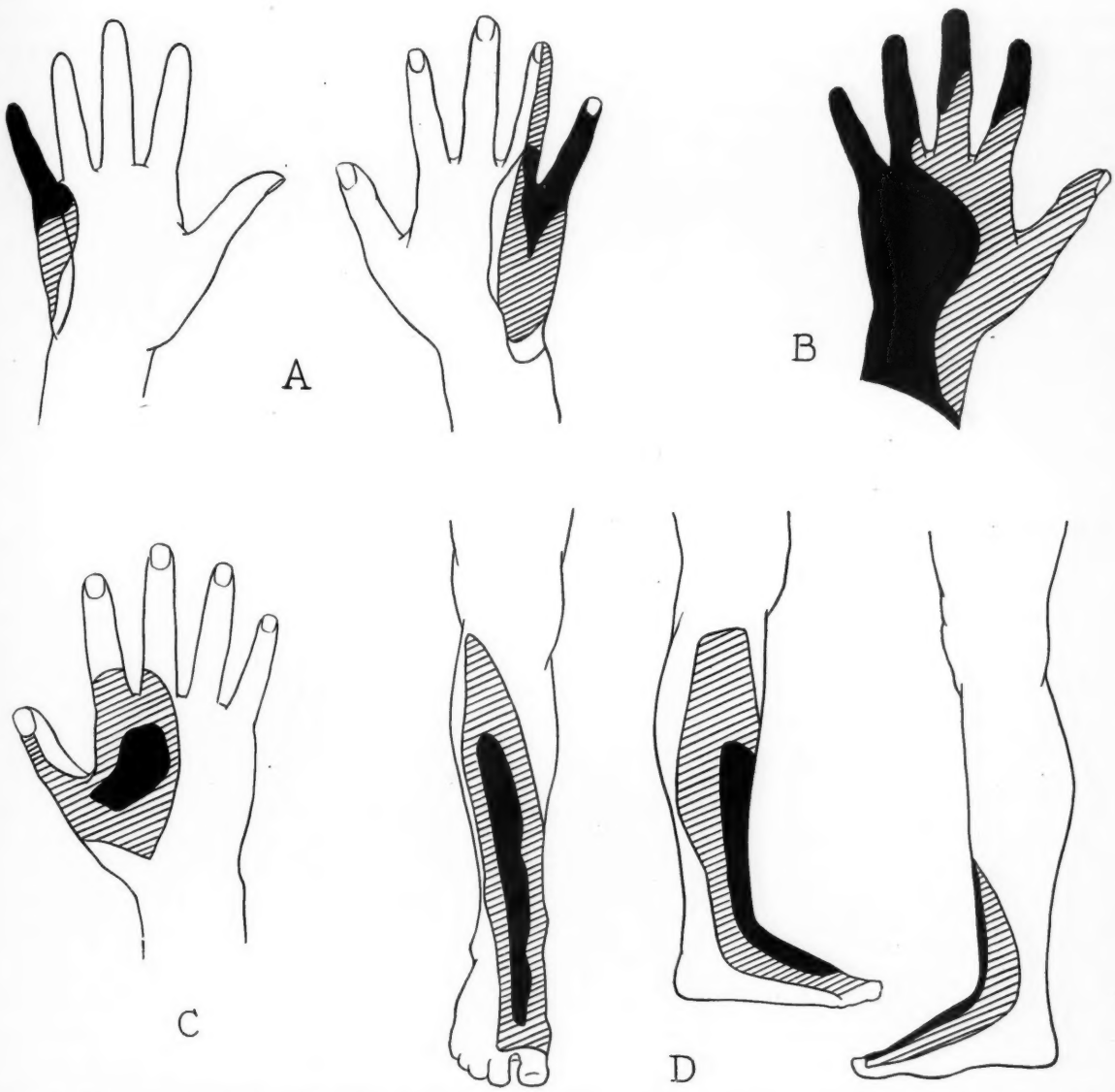

Figure 2. Sensory chart showing the pattern of return of sensation due to overlap of function of adjacent uninjured nerves.—A. Ulnar nerve.—B. Ulnar and median nerve.—C. Radial nerve.—D. Peroneal nerve.—Black, loss of pain, touch and temperature; shading, loss of touch.

supply of two nerves simultaneously injured.

Interlacing of the border of sensory loss of one type of sensation with that of another (Figs. 3 and 4).

Sensory recovery does not take place coextensively with motor recovery. In some instances, complete sensory recovery may have taken place when motor recovery was but partial. In lesions of the tibial and peroneal nerves, at times marked sensory recovery had taken place when the muscles supplied by these nerves were completely paralyzed. In ulnar nerve lesions, sensory recovery was present in 50 per cent of the cases showing motor recovery. In median nerve lesions, sensory regeneration could be demonstrated in 63 per cent of cases showing motor returns. In combined lesions of the ulnar and median nerves, sensory loss was incomplete in 88 per cent of cases while only 23 per cent of ulnar nerves showed incomplete sensory loss. Eighty per cent of recovery of radial nerves showed sensory regeneration. This was correlated more with the return of functions of the extensor longus pollicis and extensor communis digitorum than the extensors of the wrist. In injuries of the sciatic nerve, motor recovery in the peroneal part is associated more frequently with sensory regeneration in the musculocutaneous and anterior tibial nerves than in isolated lesions of the peroneal. Here the more muscles recovering the more number of cases showed sensory regeneration in the sural nerves.

Reference

1 Benisty, Mme. Ath.: "The Treatment and Repair of Nerve Lesions," *Miliary Medical Manuals*, 1918.

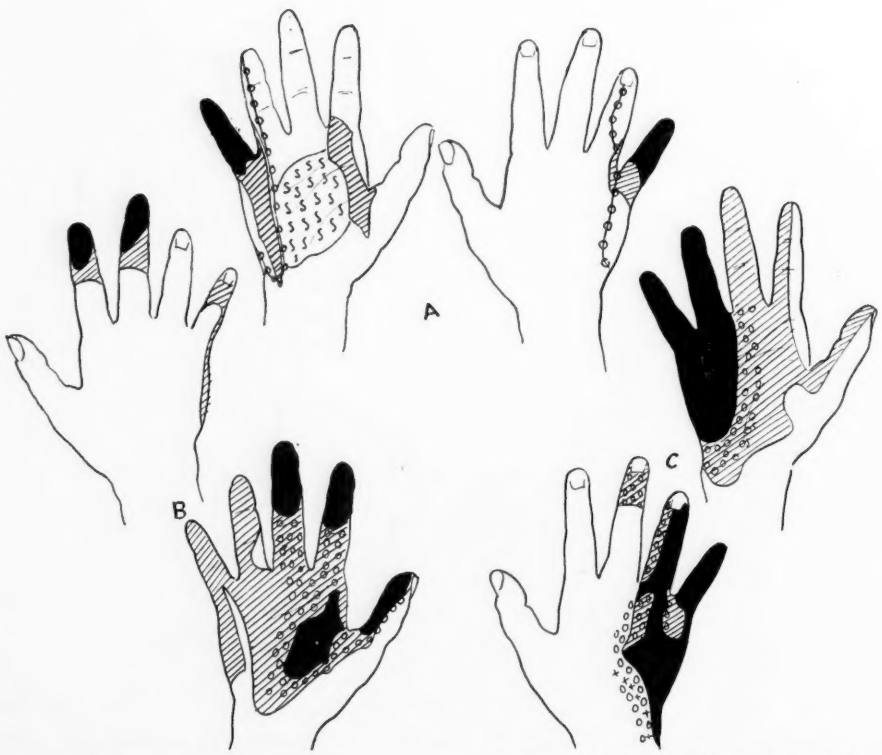

Figure 3. Ulnar and median, postoperative recovery.—A. return of sensation in area between injured nerves—B. ulnar only neurolysis, median section; suture, return of pain, not cold—C. median only neurolysis, ulnar section and suture; patchy return of pain in ulnar—Black, loss of pain, touch, and temperature; shading, loss of touch; circles, loss of temperature; letter S, hypaesthesia; letter X, analgesia.

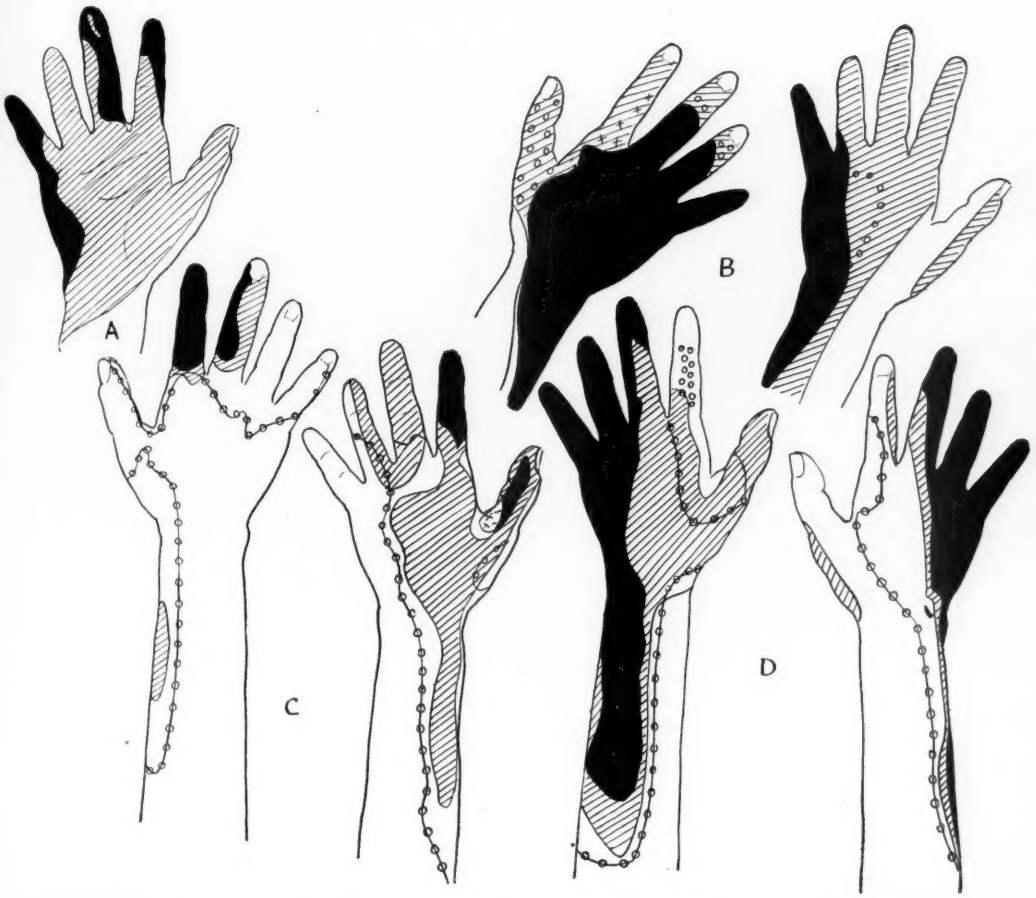

Figure 4. Brachial plexus—A. patch of return of pain in isolated supply; recovery between adjacent nerves—B. recovery of pain, not touch—C. recovery of touch, not cold; patchy return of touch—D. interlacing of loss of cold. Return of touch in isolated supply. Recovery of touch, not cold—Black, loss of pain, touch, and temperature; shading, loss of touch; circles, loss of temperature; letter X analgesia.

President of American Psychiatric Association Named to Additional Post

Dr. Edward Strecker of Philadelphia, Pa., president of the American Psychiatric Association, has been named Special Consultant to the Secretary of War for the Air Forces of the United States Army. In this capacity, Dr. Strecker will act as advisor to War Secretary Henry L. Stimson on all questions relating to psychiatry in the Air Forces. Serving as a civilian advisor, Dr. Strecker will be on call at all times by the War Department. The appointment marks the second special consultant's post for Dr. Strecker. A month ago, he was named Consultant in Psychiatry to the Surgeon General of the U. S. Navy, in which capacity he acts as advisor in psychiatry to the Navy's Bureau of Medicine and Surgery. Dr. Strecker recently established the first intensive training school for naval medical officers in Philadelphia.

Dr. Strecker has had extensive military experience. In World War I he held the rank of Major in the Army Medical Corps, as division neuropsychiatrist for the 28th Division in France. He became known as an authority on the military psychiatric disability formerly termed "shell shock."

Dr. Strecker is professor and head of the department of psychiatry at the University of Pennsylvania's Graduate School in Philadelphia, and the Medical School of the same university. He has been medical and clinical director of the Pennsylvania Hospital's Department of Nervous and Mental Diseases since 1917. He is chief of service and consultant at the Institute for Mental Hygiene of the Pennsylvania Hospital, and consultant for Bryn Mawr College and the United States Veteran's Bureau.

Author of a best-selling medical work on alcohol entitled "Alcohol—One Man's Meat," Dr. Strecker has also made special researches on abnormal behavior in children. He is staff neurologist for the Pennsylvania, Philadelphia, and Germantown Hospitals.

In 1939, Dr. Strecker was selected to deliver the Salmon Lectures on Psychiatry and Mental Hygiene. He served as clinical professor of psychiatry and mental hygiene at Yale University in New Haven, Conn., from 1926 to 1932.

As president of the American Psychiatric Association, the oldest association of medical specialists in the country, he will preside at that group's centennial anniversary next Spring in Philadelphia, site of the association's founding.

Some Observations on the Use of Curare for the Prevention of Complications in Convulsive Shock Therapy*

BYRON STEWART, M.D.
Beverly Hills, California

Before discussing the use of curare, the South American drug, which is available for experimental use in a limited number of institutions in this country, the writer would like to summarize very briefly the rapid development of convulsive shock therapy in the treatment of the mentally ill.

History

Fourteen years ago Manfred Sakel[17,18,19] instituted the use of insulin to induce shock in schizophrenic patients, after having discovered accidentally that such treatment was beneficial. Later Meduna[14] discovered that the same result could be achieved more quickly and with better control by the use of metrazol. The striking success which he reported caused the rapid acceptance of this treatment by mental institutions and neuropsychiatrists, although many of these found that their experience with the therapy was not as spectacular nor as satisfactory as they wished.

At first applied only in schizophrenia, metrazol was used later in manic and depressive psychoses. In schizophrenic states the best results were obtained in the catatonic group. Although in the beginning it seemed equally successful in all these types of mental disease, follow-up studies have revealed that a much higher percentage of relapses occur in manic and catatonic patients than in the depressive states. For this reason many physicians have discontinued the use of shock treatment with all except depressed patients, or else they have restricted its use to certain selected cases in other psychoses.

The first application of metrazol in depressive psychoses was reported in the literature in 1938.[2] Since then many investigators have reported successful results with this type of patient. Dr. A. E. Bennett[3] has reported a follow-up study of 70 cases, after 6 months to 2 years, showing that 90 per cent of these severe depressive reactions were terminated within two to three weeks of treatment with about 10 to 15 per cent of relapses. Dr. Eugene Ziskind[22] has completed a report of 38 cases of patients in the affective psychoses group of whom 82 per cent were well and 10 per cent were improved 3 to 20 months after treatment.

In 1939 Cerletti and Bini of Rome applied the use of electricity to induce convulsions of an epileptoid nature in man. This method was accepted instantly by workers in other countries and was introduced in the United States late in the same year. Due to the fact that electroshock therapy has certain definite advantages over metrazol in regard to both patient and physician it has largely replaced the latter and is now looked upon as the shock method of choice in the treatment of the mentally ill.

While neither the number of cases reported nor the time elapsed since the treatment was given is sufficient to base general conclusions upon, the results seem to show that convulsive shock therapy is indicated in depressive states, especially those occurring in the middle life, involutional or pre-senile periods. In these forms of mental illness convulsive shock therapy offers new hope in alleviating the suffering of these unfortunate individuals.

Although many reports on the use of metrazol and electroshock therapy have appeared in the medical literature, these have varied so greatly, both in observations of the patients' reaction to the treatment and in results obtained, that the conservative physician is left at somewhat of a loss as to how to appraise the procedure. There is still much controversy as to its efficacy and real worth, a controversy which arose shortly after the therapy was instituted and which cannot be definitely settled until 10 or 15 years have elapsed.

Nevertheless, the fact that the majority of reputable institutions for mental illness in the

*This term currently includes metrazol and electro shock. Recently, however, it has come to designate electro shock because the latter has largely replaced the former in modern shock technique.

United States is using this treatment at the present time makes it unnecessary for the writer to try to evaluate convulsive shock therapy in this paper.

Traumatic Complications

One of the chief reasons why many neuropsychiatrists have been so dubious about the treatment has nothing to do with its end results. They were alarmed by the problem of traumatic complications which accompanied some cases and made the procedure seem unduly hazardous, especially in the cases of aged or debilitated patients. These complications consisted mainly of spinal fractures, fractures of the extremities, dislocations and severe muscle strains, some of which might have been fractures. The incidence of serious extremity fractures of humerus or femur has been estimated by some authorities as 1.5 to 2 per cent and dislocations 17.2 per cent.[2] Compressive fractures of the spine have been reported as occurring in 43 to 51 per cent of cases.[15] Muscle soreness, especially backache, was even more frequent and in many cases persisted several weeks after completion of the treatment.

Since these complications were most serious in the very age group of patients—those in middle life or pre-senile depressive states—with which convulsive shock therapy had proved most successful, this state of affairs proved to be a contraindication to this treatment and led in some instances to its abandonment.

The prevention of complications soon began to occupy the minds of physicians. Many prophylactic measures were immediately instituted and less severe convulsant drugs were experimented with. Of the more important prophylactic measures the following were tried: Hyperextension of the spine; restraining the pelvis, hips and shoulders, and adducting the arms. These were only partially successful. Later the administration of metrazol after inducing mild insulin coma was tried in the belief that insulin produced hypotonia sufficient to prevent fracture complications. This allayed the severe anxiety of the patients so frequently noted, but added to the hazards of the treatment without preventing fractures. Spinal anesthesia was given to a few patients. The ensuing paralysis of the back and lower extremities prevented complications in these regions but failed to protect the upper extremities. In one case so treated, a fracture of the shoulder occurred.

Thus, until late in 1939 the problem of preventing traumatic complications in shock therapy remained unsolved. In September of that year Dr. A. E. Bennett began a series of experiments with curare and metrazol. This drug was reported in 1595 but its pharmacological action was not described until 1844 when Claude Bernard, the famous French physiologist, discovered that it affected the myoneural junction or motor end plates by paralyzing the synapse between nerve and muscle. Dr. Bennett's rationale for using curare in conjunction with metrazol was that it would block the neuromuscular junction and prevent excessive nerve impulses from reaching the muscles, thereby lessening the severity of the muscular contractions. Fortunately, a large amount of curare had just been brought back from South America and a quantity of this was made available for experimental research.

The results were most encouraging. According to Dr. Bennett's first report[5] made before the American Psychiatric Association in May 1940, preliminary curarization before metrazol induction entirely eliminated fractures without lessening the therapeutic effectiveness of the treatment. This conclusion was based on 122 cases, who received a total of 738 curare-metrazol shocks.

In this series only one complication was reported. One patient sustained a compression fracture of the seventh dorsal vertebra. However, in this case, because of the poor veins of the patient, the metrazol was given after curarization had worn off.

At the present time more than 100 institutions throughout the United States are using preliminary curarization before convulsive shock therapy and in many of these institutions, combined curare-metrazol or combined curare-electroshock technique has completely supplanted the use of either metrazol or electroshock therapy alone.

Before we can proceed to recommend the drug with entire confidence, however, we must take into consideration the following factors: First, the availability of the drug; second, the standardization; and third, the technique of administration.

Availability of Curare

Investigation of the availability of the drug takes us into an esoteric field. As yet no method of synthesizing curare has been discovered and our only sources of it are in the South American jungle. Curare is a gummy substance which is made by brewing the stems, leaves and roots of the curare plants or Strychnos Toxifera. The plants were erroneously classified in the strychnos family probably because other arrow poisons used in Africa and Asia were strychnine derivatives but in point of fact the action of curare is in direct antithesis to that of strychnine and in clinical medicine it has been used as an antidote. The plants are found in many regions in South America and their product is used by the Indians on their arrow tips to paralyze their game in hunting.

Curare was first reported three and a half centuries ago in Hakluyt's account of Sir Walter Raleigh's expedition to the Orinoco in 1595. At that time its manufacture was a secret known only to the witch doctors of the Indian tribes and handed down from father to son. Much of the mystery shrouding its collection and preparation by the Indians has persisted until the present time.

Its physiological action was observed by Bernard, Pelouze and Lipique, but not until 1867 was the drug applied in clinical medicine. In that year Jousset, Demine and Busch treated epilepsy, rabies, various convulsive states, chorea and strychnine poisoning with curare. Similar experimentations with the drug followed this report. Tsocanakis in 1923 treated spastic paralysis with it and Bremer in 1927 used it successfully in cases of local tetanus. Since then other workers have used it in the treatment of tetanus convulsions, causalgia and all types of spastic states. In all these conditions the action of the drug is not curative but produces a relaxing effect which gives temporary relief. The chief drawback is that the effect of curare is too transient to be utilized therapeutically in chronic spasticity.

There are more reasons why the drug has not been used extensively in clinical medicine. It has been impossible to obtain for clinical use in large quantities; it has varied greatly in its potency, no one sample comparing in strength with another; and there exists a strong prejudice against the clinical application of the drug because of its reputation as a mysterious and deadly poison.

The first of these difficulties was solved by Richard C. Gill,[10] an American living in Ecuador, who in his recent book, White Water and Black Magic, describes how he became interested in certain plant drugs found in the South American jungle, particularly curare. He organized a field expedition in 1938 and after many months spent among the Indians collecting the drug, he returned to this country with thirty pounds of tested curare, the largest amount ever brought back for research purposes.

The task of refining and standardizing this supply of the drug for clinical use was undertaken by E. R. Squibb & Sons, whose medical research department worked in cooperation with Dr. A. R. McIntyre of the University of Nebraska. It is due to their careful work that for the first time since the physiological action of the drug was discovered, a pure, standardized product for intravenous use has been made available in large quantities for therapeutic experimentation. The distribution of the drug was limited for an experimental period to prevent its too indiscriminate and enthusiastic reception, before the technique was thoroughly tested.

Standardization

In the standardization of curare, an infusion or alcoholic extract of the drug is prepared. A satisfactory method of assay is the head-drop method in the rabbit. The technique is as follows: The curare solution is injected slowly into the ear vein of a rabbit, lying face down on a board. That amount is injected which, after two and a half to three minutes, will cause sufficient flaccidity of the neck muscles to prevent the animal from holding its head up. This is the so-called physiological end-point. It is clear-cut and similar to that seen in human beings. From the amount of curare solution per kilo required to produce the end-point in rabbits, the amount per kilo needed to produce a similar effect in humans is calculated. The dose is estimated according to the body weight of the patient. The drug is remarkably stable and the standardized solution will retain its strength indefinitely. The preparation is not destroyed by autoclaving. It is recommended

that the solution be kept in a refrigerator, as room temperature may cause the formation of a precipitate.

Action of Curare

The physiologic action of curare is upon the motor end plate or synaptic junction. Acetylcholine is the chemical mediator at the synaptic junction and facilitates the transmission of the nerve impulse from nerve to muscle. Curare inhibits the action of acetylcholine and thus prevents the full effect of the normal, physiological nerve stimulus from reaching the muscle. This mitigates the force of the convulsive seizure and largely eliminates the violent contractions observed in a typical metrazol convulsion.

The action of curare is selective. It acts upon all striated or voluntary muscle but first affects the short muscles of the eyes and throat and later the muscles of the head, neck, extremities, intercostals and diaphragm. The heart is not directly affected and sensory nerve functions remain intact.

The foregoing effects are noted after intravenous injections of the drug. There is no absorption when administered by mouth. The physiological effects noted shortly after intravenous injections wear off very rapidly and the drug appears to be completely eliminated within ten to fifteen minutes. It is believed to be acted upon by the liver and excreted by the kidneys. In lethal doses death occurs from asphyxia due to respiratory paralysis. The so-called lethal dose, however, is variable. Fifty times the amount that would ordinarily produce death has been given to dogs without fatality by maintaining artificial respiration. The rate of absorption has a great deal to do with lethality. In animals it has been shown that large amounts can be given if the drug is injected slowly. It has been estimated that the amount of the drug necessary to produce curarization in a human being is about 65 to 70 per cent of the lethal dose.

Recent animal experimentation has shown that a second dose of curare given within one to five hours after the initial dose is more effective than the first dose, even if the second dose is less than the first. This would indicate that there is considerable prolongation of effect of the active curare even though the clinical signs of curarization may have disappeared. This experimental reaction has also been known to occur in man. A subject received 70 mgs. of the drug and later failed to show any curare-like effect. After an hour an additional 55 mgs. of curare were given and this was followed by metrazol. After the convulsion the patient showed signs of malaise, complete relaxation and finally respiratory parlysis. The pulse remained good but it was necessary to administer artificial respiration and antidotes for a period of four minutes before spontaneous respiration was resumed. In view of these observations, it should always be borne in mind that curare should not be repeated within twenty-four hours.

Method of Administration with Clinical Signs and Symptoms Commonly Observed

The most suitable preparation of curare is that called Intocostrin, prepared by E. R. Squibb & Sons, and now available for clinical use. The solution is aqueous in nature and is so standardized that 1 cc. of the solution represents 20 mgs. of active curare principle. In clinical practice it has been determined that 20 mgs. will curarize 35 to 40 pounds of body weight. From this determination the individual dosage can be computed. Females take less than males; younger patients less than older patients.[5] To allow for individual variations in reaction, I have been using 10 mgs. less than the above estimated dose in the initial treatment of each new case. Even though complete curarization may not be obtained by this procedure, there is sufficient effect of the drug to prevent complications and at the same time the risk of respiratory embarrassment is obviated. If in the first treatment curarization is not complete, I increase the amount of the initial dosage for subsequent treatments by 5 mgs. until the desired stage of curarization is reached. I estimate this stage to be when the patient can barely lift his head. There appears to be no increased tolerance to the drug during the course of treatment.

The estimated dose is given intravenously in a period of from one to one and a half minutes. The time element is important because the effect of the drug is largely dependent on the rate of injection. Therefore it should be stressed that one to one and a half minutes is the shortest length of time in which curare can be injected with safety.

The signs and symptoms noted during the

injection and appearing in the following order have been accurately described by Bennett: "The patient first complains of haziness or fuzziness of vision. Next, bilateral ptosis appears with slight nystagmoid movements, relaxation of the face and heaviness, with relaxation of the jaws. The patient at this point complains of tightness of the throat and huskiness of the voice. Generalized heaviness and weakness of the neck muscles with inability to raise the head are followed by weakness to complete paresis of the spinal muscles, legs and arms. Lastly, shallowness of respiration appears due to weakness of the intercostals and the diaphragm." The writer has observed all of these phenomena except complete paresis of the arms and legs.

The maximum effect of curare is reached approximately 2 to 2½ minutes after completion of the injection. At this point it is usually very difficult for the patient to lift his head because of the partial to complete flaccidity of the neck muscles. (During the injection this test is carried out repeatedly so that the course of the paralysis can be controlled.) When the patient has reached this stage, referred to as "complete curarization," the convulsion is induced.

The difference in the convulsion induced by electroshock alone and that induced by combined electroshock and curare is striking. The severe tonic rigidity and clonic jerkings which characterize the electroshock convulsion are greatly reduced in intensity through curarization. Opisthotonos does not occur. The curare-controlled convulsion is so much milder that precautionary measures, such as hyperextension of the spine, tongue gag and restraint of the body are not necessary. Although the factor of fear has been largely eliminated by the use of electroshock it has been observed that patients do show less apprehension when curare is used. The writer has the impression that this is due to the relaxing effect which the drug gives.

Because of the action of curare upon the respiratory muscles, the patient should be carefully watched after the convulsion has ceased and before consciousness has returned. Artificial respiration, epinephrine and prostigmin should be administered if there is evidence of respiratory embarrassment. Experience to date with the drug indicates that this does not occur when the proper technique has been followed but the physician should be prepared to deal with any emergency connected with respiratory function. Bennett speaks of holding the patient's relaxed jaw forward if the tongue or excessive saliva interferes with breathing and also refers to the possibility of the necessity for intubation. No case has been reported as requiring the latter procedure, however. In fact, the effect of the curare wears off so rapidly that the need for the precautions mentioned above is practically over before consciousness returns.

The care of patients following the combined curare-electroshock treatment is easier because there is less discomfort, restlessness, headache, nausea and muscular soreness than there is following the electroshock convulsion. The diminishing of these discomforts greatly simplifies the after care of patients and constitutes one of the chief advantages of curare.

During the past two years the writer has given 845 curare-controlled convulsive shock treatments (193 curare-metrazol and 652 curare-electroshock) to 108 patients with no fractures. At first curare was used with a few selected patients, but as its efficacy was observed its use was extended to all convulsive shock cases.

The patients treated represent both sexes with ages ranging from 18 to 70. In this series of cases only one instance of respiratory paralysis occurred, which was quickly overcome by artificial respiration. It is the writer's belief that this could have been avoided by the procedure of reducing the estimated initial dose by 10 mgs., as described earlier in this paper.

Twenty-two of the cases or 20.3 per cent were over 60 years of age. One patient showed myocardial damage with coronary artery disease. The treatment was given successfully to these cases without the feeling that undue risk was being taken. Before the advent of curare it was considered unwise and a risk to give convulsive shock therapy to aged patients and to those with accompanying organic disease. Curare, therefore, may open a new field in shock therapy by enabling the conservative physician to extend its use to those patients in the older age groups and to many of those who, because of various physical disabilities were heretofore denied this beneficial treatment.

Conclusions

Research in shock therapy has progressed along two lines. The first of these had as its aim the finding of a short and successful treatment for some kinds of mental illness. The purpose of the second was to make this treatment less rigorous and to insure the safety and comfort of the patient. Curare may be said to be the most important single advance yet made in this direction, since it has eliminated or greatly reduced those visible and immediate hazards which include fractures, dislocations, and all forms of post-convulsive discomfort. It is difficult to see how any drug could be more suitable for this purpose than curare because of the ease of administration and the fact that the periods of action of electroshock and curare coincide almost ideally. At least it meets the existent need better than any other procedure yet evolved.

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Cerebral Anoxia*

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Cerebral anoxia has always been of prime importance. It has been involved in industrial medicine, particularly in coal mining, where asphyxia may result from cave-in or from liberation of methane or carbon monoxide. It is the vital mechanism in drowning or strangulation. In recent years its occurrence under nitrous oxide anaesthesia[1,2,3,4] has been noted with increasing frequence. In modern warfare suffocation may be the prime factor in casualties occurring in fire or collapse of buildings. With the development of flying at high altitudes, cerebral anoxia has taken a place of even greater significance, and for every miner who is overcome with methane there may in the future be many individuals who succumb to failure of inhalation apparatus in rarified atmosphere.

The following discussion is based largely on case material of the Western State Hospital. One case has been reported in detail previously.[5]

Etiology

It is ever more apparent that many different asphyxial causes or agents produce essentially the same clinical and pathological effect through a common medium: cerebral anoxia. It is doubtful that any agent asserts a direct asphyxial influence, on nervous tissues, of a nature peculiar to that agent. For example, carbon monoxide has an affinity, surpassing that of oxygen, for hemoglobin (but is not in itself a specific poison for nervous tissue), and failure of inhalation apparatus at high altitudes may expose one to a rarified atmosphere, but the end result in each case is the same: low concentration of oxygen in the blood, hence, in the tissues of the central nervous system. It is true that nitrous oxide and barbiturates[6] have specific anaesthetic or soporiphic properties, but when asphyxia occurs from these two or from other similar agents, it is because the percentage of oxygen in a mixture of gases inhaled is, or because, as a result of cardiac or respiratory depression, the blood passing to the brain circulates too sluggishly or is in a state of insufficient oxygenation.

The actual pathophysiologic mechanism by which anoxia affects nerve tissue has been the subject of considerable speculation. Such phrases as, "special sensitivity of cells for anoxia," are of little real assistance. Alexander[7] found in cases of carbon monoxide poisoning that the areas of softening in the globus pallidus corresponded to those visualized in "normal" specimens by injection of a suspension of india ink into the arteria choroidea anterior. This vessel, found occluded in the aforementioned cases, was considered to be relatively susceptible to thrombosis because of the high ratio of its unanastamosing interval to its diameter. The long thin venules of the cerebral white matter were likewise found thrombosed.

No inquiry into the vascular occlusion theory of pathogenesis of necrosis in asphyxia is complete without recognition of the almost unfailing predilection of cerebral infarction, due to arteriosclerotic thrombosis, for the putamen; this phenomenon is of particular interest when one recalls that the aforementioned arteriosclerotic thromboses are thought often to result from a drop in intracerebral blood pressure, in the absence of any special degree or situation of arteriosclerosis differing from that found in other cerebral vessels of the same individual, but in the presence of vascular architectural peculiarities found regularly in all individuals.

In a case of anoxia from strangulation with a survival interval of eighty hours,[5] histologic study revealed ischemic necrosis throughout the brain of nerve cells and of their processes, with predilection for the cells of the globus pallidus; necrosis of walls of blood vessels was seen in abundance, but no gross or microscopic thromboses were found.

Most of the important causes of cerebral anoxia have been mentioned previously in this discussion. Among others, two additional

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causes of unusual interest are: tracheal collapse from postoperative peritracheal hemorrhage, and pulmonary embolism.

Symptoms

The signs and symptoms of cerebral anoxia depend upon the severity, suddenness of onset and duration of the anoxia.

Whether chronic anoxia is a source of cerebral dysfunction has been doubted by some. Krannenburg,[10] however, found in garages in which the air contained 0.2 per cent carbon monoxide that almost all the workers complained of headache. A woman receiving treatment for pernicious anemia recently volunteered that she felt much more alert mentally than previously, following an increase in the percentage of hemoglobin in the blood.[11]

Thorner and Lewy[12] found ischemic changes in the brains of guinea pigs and cats subjected to repeated experimental anoxia, high atmosphere being reproduced in the pressure chamber. They suggested the possibility of a gradual lowering of the cerebral reserve in individuals repeatedly exposed to low oxygen environment. Of interest in this regard is the fatigue syndrome, in flyers, well known to aviation physicians as aero-neurosis. A chronic fatigue state of this type, of course, results in part from nervous and physical strain; however, repeated exposure to rarified atmosphere may play a part. Adequate oxygen therapy eliminates the element of anoxia from the aforementioned fatigue syndrome.[13]

The symptoms of acute anoxia are characteristic and respond dramatically to oxygen therapy. At the outset, judgment and perception may be improved. The tragic consequence of such an occurrence while flying requires no comment. There may follow vertigo, weakness, neuralgia, somnolence and coma. Cyanosis may be marked, dimming of vision is common, and the blackout of the power dive is a manifestation of cerebral anoxia. Loss of sphincter control and convulsions may occur. The latter is well illustrated experimentally in rabbits[5] whose cervical regions are compressed; clonic convulsions appear after three minutes, lasting about thirty seconds. The precipitation of convulsions, during insulin hypoglycemic state, with nitrogen inhalation is based on anoxia.

One of the insidious things about cerebral anoxia, depending on the duration and severity of the exposure, is that when the individual survives, damage to nerve tissue may have occurred which will result in a great variety of persistent symptoms. The picture may be that of involvement of the nervous system at any level and may simulate any lesion from Korsakov's psychosis to peripheral motor and sensory neuritis. It may give memory defect, emotional disturbance and loss of a sense of responsibility. The presenting symptoms may include headache, weakness, confusion, excitement and hallucinations. There may be paresis, dysarthria, tremor and choreiform movements. The syndrome may resemble more or less Wilson's disease, Huntington's chorea or Parkinsonism. If the spinal cord is involved, multiple sclerosis or Landry's paralysis may be simulated. The peripheral neuritis in the chronic form of poisoning is in no way specific in character.

Illustrative case: A man, aged 28, was admitted to the Western State Hospital June 5, 1942. Prior to his asphyxia he was said to have been intelligent, emotionally stable and in good general health. May 2, he had attempted suicide with carbon monoxide inhalation, but was found and taken to a hospital in a comatose condition. He gradually recovered and in several days was able to be on his feet. He had a complete change of personality, becoming noisy, uncooperative, silly, confused, childish, disorientated, and was mentally retarded. There was an extreme memory defect; the inability to remember brought tears to the patient's eyes. There were marked choreo-athetoid movements of the head and all four extremities, with frequent grimacing. Deep reflexes were hyperactive, with questionable responses to the Babinski test. There was a marked intention tremor which made such acts as lighting a cigarette difficult. Encephalographic study revealed no abnormality of the cortical or ventricular outline.

Pathology

Clinical pathology: Changes in the spinal fluid are relatively mild and are not characteristic. There is frequently an elevation of pressure and there may be a slight increase of globulin and total protein. More uncommonly, there may be a mild lymphocytosis and a low grade reaction may occur with colloidal gold.

Pathologic anatomy: When anoxia is rela-

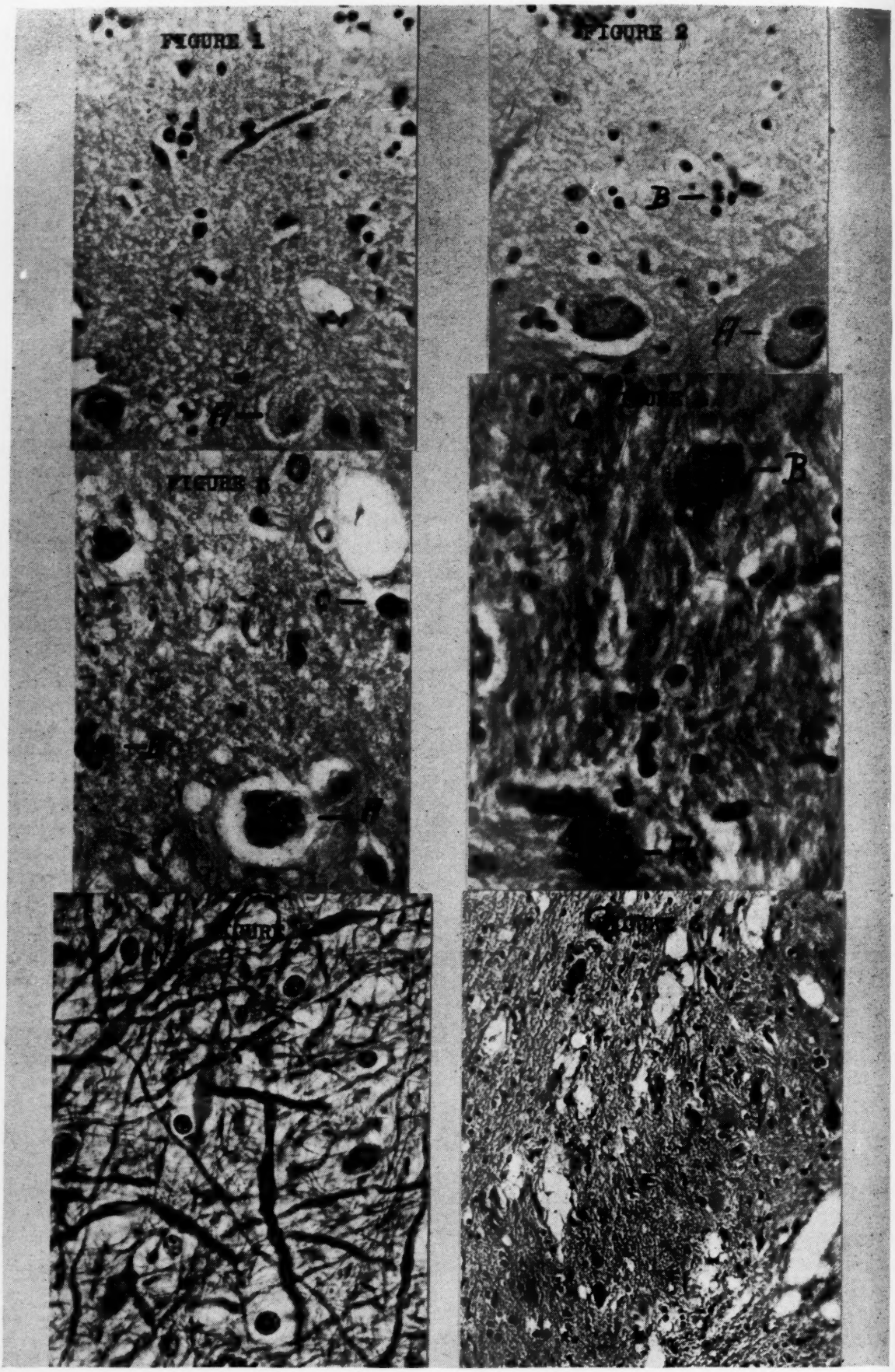
FIGURE 1
FIGURE 2
B
B
FIGURE 3
C
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B

tively mild, dysfunction of nervous tissue may occur in the absence of any demonstrable histologic change, regardless of rapidity of fixation of tissue or variety of staining methods.

When the anoxia is more severe, pathologic lesions begin to make their appearance. Nerve cells may be seen in a state of chromatolysis (Figs. 1, 2) with eccentric nuclei, rounded swelling of the cell bodies and peripheral dispersion of ill-defined Nissl substance; there may be proliferation of satellitic oligodendrocytes about the nerve cells, and some slight degree of early perivascular infiltration may be seen. Congestion as a rule is present, associated with edema, the latter giving the tissue a spongy appearance, and petechial hemorrhages are not uncommon. These changes, at least to some extent, may be considered reversible.

Following severe asphyxia the gross and microscopic changes depend not only on the duration and degree of anoxia but also on the length of the post-anoxial interval, during which anatomic changes may take place. Grossly there may be either cystic cavitation or spongy glial scarring of each globus pallidus. There may be scattered foci, usually minute in size, of degeneration of the cerebral cortex and to a less degree of the white matter. In time these become relatively firm and white. Microscopically, profound changes are seen throughout the brain substance. Relatively early there is disintegration of the nerve cells (Fig. 3). These commonly have a marked foamy appearance, lipoidal vaccuoles being strongly outlined in silver preparations. There is a diffuse spongy softening of both cortex and white matter, and satellitosis and perivascular cuffing may be severe. Later, nerve cells affected either disappear entirely, leaving a cluster of satellitic oligodendrocytes or become shrunken and pyknotic. This process reaches its maximum in the globus pallidus (Fig. 4) where nerve cells are dense, homogeneous purple masses, having no neurofibrillar or other recognizable structural detail. In the cerebral cortex, layers III and IV appear relatively susceptible; this is difficult to explain adequately. Small blood vessels may undergo severe degeneration with loss of detail; the end result of this may be calcification, which, in the globus pallidus, is a suggestive, but not unmistakable, sign that asphyxia has occurred at some time or another. It may occur as a senile or arteriosclerotic lesion, and must be distinguished, with special stains, from iron. There is a rather intense proliferation of glia. Axones are beaded, tortuous and fibrillated (Fig. 5). Proliferation of some vessels may be active and in foci of previous necrosis one may see, later on, scars consisting of masses of astrocytes intermingled with new-formed blood vessels whose walls are rather cellular. The aforementioned changes are found throughout the cerebrum and form a reasonable pathologic basis for the occurrence of mental, motor, and sensory symptoms. They are seen also in the olivary and dentate (Fig. 6) nuclei and in the cerebellar cortex; this will explain ataxic phenomena. They are most profound in the lenticular nuclei and, as we have shown, symptoms of basal ganglion injury may be marked.

LEGENDS

Figure 1. Motor cortex. Chromatolysis of nerve cells (A), satellitosis, congention and edema. H & E x 480.

Figure 2. Globus pallidus. Chromatolysis (A), satellitosis (B). H & E x 480.

Figure 3. Motor cortex. Disintegration of nerve cells (A), disappearance of nerve cells leaving satellitic cluster (B), pyknosis (C), degenerative rarefaction of tissue, edema. H & E x 600.

Figure 4. Globus pallidus. Extreme pyknosis (A), cell disintegration with satellitosis (B), proliferation of glia, degeneration of axones. H & E x 720.

Figure 5. Motor white matter. Beading, fibrillation and tortuosity of axones. Bodian (silver protein) x 660.

Figure 6. Dentate nucleus. Degenerative changes of nerve cells and of intercellular tissue. H & E x 180.

Summary

Cerebral anoxia, always vital, is becoming increasingly important with the development of flight at high altitudes. Various causes of cerebral anoxia have the same clinical, pathologic and physiologic effect. This effect depends on the severity and duration of anoxia. Cerebral anoxia may produce a number of mental and neurologic signs and symptoms. At first there may be no recognizable pathologic change; later, chromatolysis and other similar associated changes may occur which may be considered reversible. If anoxia is severe and prolonged, irreparable brain damage will occur, from which there is little hope of recovery. Residual symptoms may indicate involvement of peripheral nerves, spinal cord, basal nuclei, or cerebral or cere-

bellar structures. Pathologic changes in such cases may consist grossly of necrosis of the lenticular nuclei and also of small foci of degeneration of the cerebral cortex and white matter. Nerve cells disappear or become pyknotic. There may be proliferation of glia and blood vessels. Axones may degenerate. The microscopic changes are pronounced throughout the brain. They are most severe in the basal nuclei; and they may be marked in the olivo-cerebellar system.

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A Study of 50 Women Patients Hospitalized for Alcohol Addiction*

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This paper is a review of the case histories of 50 women patients, addicted to alcohol, who were admitted for treatment at the New York Hospital—Westchester Division during the six year period September 1934 to September 1940. In part it complements Wall's[1] study of alcoholism in women who were treated at the same hospital between 1920 and 1933. It is also an attempt to evaluate the role a mental hospital plays in the solution of a person's drinking problems and in her subsequent life adjustment.

The study includes cases of alcoholism in all the diagnostic classifications. They are proportioned as follows:

Alcoholism without psychosis	30
Psychopathic personality with alcoholism	2
Delirium tremens	2
Korsakov's syndrome	6
Acute hallucinosis	5
Alcoholic deterioration	4
Alcoholic psychoses—other types (involutional psychosis with alcoholism)	1
Total	50

As the above figures show, 20 of the 50 patients had their addiction to alcohol complicated by psychotic manifestations.

The family histories of our patients reveal a rather high incidence of maladjustment among the fathers. Fifteen (30%) were addicted to alcohol. An additional five were either psychoneurotic to a serious degree or psychotic. Success in business and prominence in their social circles were enjoyed by a majority of the fathers (34). Their influence at home has been very difficult to evaluate in most instances. Thirteen fathers had strongly domineering personalities, and in eight families the father had an obvious, strong attachment to the patient.

The mothers as a group were considerably better adjusted than their husbands. Three were addicted to alcohol and a total of seven were handicapped at some time by either alcoholism or a frank mental illness. Most mothers (35) were responsible, and affectionate. Marked indifference and irresponsibility were

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noted in eight cases. Domineering and overprotective mothers were few and evidence of mother-daughter attachments or antagonisms were rare.

The home situation during the patient's childhood was quite happy in most cases. It could be considered unhappy or deleterious through incompatibility of parents, or absence of a parent, in 12 instances. A survey of sibling relationships showed that the patient was the youngest in the family (2-6 siblings) in 19 instances and the oldest in nine. About one-half, then, had intermediate positions on the age scale. In only one instance was the patient an only child. Alcoholism or mental illness among the siblings occurred in 14 cases. Evidence of significant attachment or jealousy was slight.

The clue to the drinking problem probably will be found among the patient's personality traits manifested prior to the period of alcohol addiction. The methods of appraising the personality structure vary with the investigator. Since these appraisals stem from anamneses that are at best approximations of the true pictures, it is not surprising that the literature is filled with contradictions.[2,3] In this study we have attempted to evaluate the patient's functional adjustment in the various fields that as a group constitute our present-day environment: the family, society, self, occupation, and sexual life; also to be noted are the patient's intellectual capacities, health and disposition. It is felt by some[2] that there is a rather marked difference in personality between those alcohol addicts who show psychotic elements and those who do not. Our group of patients fails to bear this out, and we therefore feel justified in considering all our patients as one group. Nor does there appear to be any reason for separating those suffering from alcoholism with psychosis from those without psychoses on a basis of precipitating factors, duration of illness, or prognosis.

The data covering the periods of childhood and adolescence indicate that the great majority of the patients were adequately endowed intellectually, enjoyed good health, and lived in comfortable, favorable surroundings, relatively free of crippling parental attachments. There were very few psychopathic traits or significant disturbances in the adjustments made by the group as a whole during this early life period.

The data on the adult years were gathered with especial care that only pre-alcoholic personality characteristics be included. Most of the patients were housewives (33). Five were business women, seven actresses, and five unemployed. In religious preference 41 were Protestants; nine were Roman Catholics. There were no Jewesses. The economic status of the group as a whole was quite high. There were only four instances of significant economic insecurity. A very large majority had made good ambivert or extravert adjustments for some years. Energies, interests, and social activities were at an average level or better. But while the group was making an apparently good adjustment, only a few (nine) were satisfied with the patterns their lives were assuming. Vague dissatisfaction or even openly expressed active dissatisfaction, is to be noted in a large majority. The reasons for this are not indicated in many of our histories, but a few factors should be pointed out. Psychosexual development was considered satisfactory in only twenty instances. There were five women who were unmarried, 21 who were immature, self-indulgent, narcissistic and four who were apparently arrested at a homosexual level. Only one was overtly homosexual. A few others found their adult adjustments difficult because they had received over-indulgences through most of their early years, and continued to demand them, eventually unsuccessfully. Physical illness was a handicap in ten instances.

The reasons for excessive alcoholic indulgence as given by the patients or their relatives are numerous. They may occur singly or in combination. The ill-defined dissatisfaction with life, as above noted, is rather frequently mentioned as a cause for drinking. More concrete matters are: loss of a close relative (ten instances); a marital situation difficult for an immature, insecure individual (19 instances); strong involutional factors (16 instances); threatening or actual economic insecurity (7). Social drinking was considered highly contributory to alcoholism in 22 instances. A strongly significant factor in 30 cases was the presence of rather marked depressive or anxiety feelings prior to drinking.

The course of the average patient from her first social drinking to the hospital covers a period of twenty years. Social drinking in all

cases but two started at about the age of twenty. When drinking became heavy because of probable personality difficulties, the average age of the patients was 30 (50% were in their middle twenties). Drinking became incapacitating when average age was 35, and on admission the age had risen to 40.

Treatment

When the patients were admitted to the hospital, it was planned that treatment would cover a prolonged period. Thirty had had hospitalization elsewhere, usually for brief periods. Fifteen patients sought admission voluntarily. Thirty-five were admitted on a committed status. The matter of commitment was usually taken up in great detail with the patient and her family before beginning treatment. As a rule the patient petitioned for her own commitment, preferably for a period of one year. This was not true in all instances. Neither did this mean that the patient was to remain in the hospital during the entire period of commitment, but at least the patient and family were assured of supervision for this period.

The patients were examined by their personal physicians and by consultants in internal medicine, gynecology, otolaryngology, ophtholmology, and dentistry. Laboratory studies were complete. Efforts were then directed toward depleted physical conditions and toward those organic mental changes that presented themselves. The regime included rest, nourishment, vitamins, prolonged baths, massage, hydrotherapy. When the physical condition permitted, the patients were placed on a rather full schedule of physical education, occupational therapy and social activities, as directed by the physicians. Psychiatric interviews were held at regular intervals in an effort to induce the patient to discuss her life situation and problems. As will be pointed out below, this particular feature has proved both very important and difficult.

The most outstanding feature of the hospitalization of this group of patients was the marked resistance to facing the problem of alcoholism. Thirty remained in the hospital less than three months; 17 left within four weeks. Those on voluntary status stayed in the hospital on an average of two or three months. Those on committed status remained an average of four and a half months. Although most patients (38) cooperated fully in hospital routine, only 16 participated in interviews in an effort to understand their chief problems. The other 34 resisted the psychotherapeutic approach either in a gracious evasive manner or with open antagonism. On leaving the hospital those who seemed to have gained some insight to their difficulties numbered 14. Twenty-six were strongly antagonistic. With rare exceptions, all patients returned to unmodified home or occupation situations.

Results of Treatment

All patients have been living apart from this hospital for at least one year. Reports on 46 have been received from time to time from relatives of the patients and from hospitals at which the patients have been treated subsequently to their discharge from this hospital.

Those patients who abstained from alcohol for one year or more numbered five. Two of these resumed light drinking without incapacitating effects. Four other patients modified their drinking to a point where their excesses were less disastrous and further hospitalization was not required. The rest, 37, resumed pathological drinking within one year. Over two-thirds of this group resumed drinking within three months. These figures, however, do not tell the whole story of the role of a mental hospital in the management of alcoholism. Difficult to evluate, but still important, is the modification of the attitudes of the patient's family, and the subsequent improvement in the family management of the patient during her excesses.

The nine patients who were aided in their alcoholic problems by the hospital regime were, with one exception, fully cooperative in psychotherapeutic interviews as well as in the hospital routine. This group, then, stands out in sharp contrast to the great majority of the patients who were resistive to the psychiatric approach. The one exception, as will be pointed out below, was a woman suffering from a primary depressive illness who took alcohol to incapacitating degrees in reaction to the depression. When the depression passed, alcoholism ceased.

Sketches of this case and three others who abstained from alcohol for one year or more are presented. They illustrate the diversity of personality types and the various problems

found among the alcohol addicts.

Mrs. L. K., age 54, had been drinking very heavily for about three years. Mental illnesses were prominent in both ancestral lines and two of the patient's siblings suffered from chronic dementia praecox. Two other siblings are very seriously addicted to alcohol. The patient was the second of the five siblings. Her childhood development was not remarkable. Schooling and health were satisfactory. Eventually she became a successful corsetiere and married at 32. She had two children and the home life went smoothly. There was a tendency, however, to prudishness, oversolicitude, and rigidity in household management. When 51 her husband became an invalid and the patient became somewhat depressed. She turned against her children. Menopausal symptoms became manifest and patient started drinking. The amounts consumed were prodigious. She drank alone, became paranoid, had many angry outbursts and had about eight different periods of hospitalization for brief periods. Auditory and visual hallucinations developed and patient was admitted to this hospital. Physically she demonstrated a marked peripheral neuritis and an enlarged liver. She received appropriate medication, participated in a short while in routine program matters, but evaded all attempts to discuss her life problems. The patient's psychosis was a mixed one that was considered best classed as Alcoholic Psychosis —Korsakov Syndrome. After nine months in the hospital on committed status the patient appeared quite comfortable but clung to some delusional ideas, and had occasional temper tantrums. Very gradually she felt more self-assured and on leaving the hospital adjusted well. Recently, two years after discharge, the patient lost her husband through death. She drank on two occasions, but not to incapacitating degrees. She is now quite comfortable, drinking an occasional social glass without desire for more.

Mrs. G. H., age 31, had been very dissatisfied, bored and drank heavily for three years. Her father, a successful business man, had been a strict discplinarian, selfish, quick tempered. The mother, vain, impractical, extravagant, divorced her husband and alone raised the patient from the age of six. The maternal grandfather had been addicted to alcohol. The patient, an unwanted and only child of an unhappy marriage, was extremely indulged and overprotected throughout childhood. School was sporadic and whatever interests she may have had in such matters as art, theatricals and music were very short lived. She could not get along with her teachers and found making friends very difficult. She failed as a movie actress after six unhappy years of attempts; she failed to hold a married lover because of her indifference to intercourse; and when her mother died she turned to a shy temperamental man, who though never her husband legally, protected her off and on for the next ten years. Life became a matter of boredom, horse racing, drinking, and love affairs with poets. After a mild depression she attempted to live alone, failed again to gain theatrical work, and drinking reached a point of one pint of liquor per day. Poverty began to complicate increasing loss of interests and increasing difficulty in associating with people. In a short while, increased drinking led to neurotic symptoms, partial blindness, gastric symptoms, auditory hallucinations, and finally a terrified confusion. A few weeks after admission patient's illness was classified as "alcoholic psychosis—acute hallucinosis." After she gained composure she entered into all hospital routine with great enthusiasm and tried to work over with her physician concrete plans for the future. After three months of hospitalization, she demonstrated considerable insight in her problem, and on leaving entered a school of dress designing. Occupied with this and home study, she also managed a home for herself and her recently married husband. For two years the patient has abstained from alcohol, and has been fairly comfortable in spite of a persistent tendency to avoid people.

Mrs. G. P., age 44, had been drinking alone for four years to ease her worries. While intoxicated she often expressed the wish to die. The father was a prosperous financier, sociable, religious and liberal. The mother was an obstinate, dominating person. All the siblings were making very precarious adjustments; one died of alcoholism and a second frequently drinks to excess. The patient, the second of six siblings, had a healthy, happy childhood, finished a very satisfactory education at 18, travelled, and enjoyed considerable popularity with boys. She tended to be quiet, conventional, sensitive and on rare occasions

exhibited temper tantrums when hurt. At 25 she married a worrisome, opinionated man who was dependent on his father and who attempted to dominate the patient. She also had to contribute to his support and tended to resent this. Sex adjustment was satisfactory. She had two children after medical measures to combat sterility. Drinking started shortly after marriage, and gradually increased for about 12 years until it became unmanageable at one pint a day four years before admission. She had become withdrawn socially and was very dependent upon her husband. On admission on committed status she was depressed, irritable, resentful and expressed guilty feelings and suicidal wishes. She spoke of wanting her husband to be as successful as her own father. Soon she was in excellent physical condition. Her attitude became friendly, and about her alcoholism she felt very guilty and ashamed. Participation in psychiatric interviews was excellent. After seven months she left the hospital because of husband's financial embarrassment. Relations with him became progressively more difficult and patient had to assume all responsibility for the family. The husband left after three years to live with his mother, and patient had one or two extramarital affairs about which she had some misgivings. She never returned to alcohol and regarded her former alcoholism as stupid.

Miss V. W., aged 26 on admission, had had periods of uncontrolled drinking for at least six years. Her father, a wealthy mid-west banker, was hypochondrical and addicted to alcohol. The mother, a dominating, rigid woman, suffered a post-partum psychosis. In her early years the patient was unhappy, antagonistic, and given to temper tantrums. Though intelligent she was so mischievous and rebellious she was finally forced to leave school at 16. At 18 she tried dramatics in New York City with moderate success and at this time fell in love with a series of older men. When they repeatedly left her she started drinking. Socially she was very popular and easily gained the sympathy of others. She complained of deep feelings of inferiority. Her sexual excesses resulted in considerable trouble. A particularly distressing love affair ended in delirium tremens. During the next two years unhappy affairs with older men and drinking continued and she was hospitalized four times for brief intervals. At times she worked as a salesgirl. For the six months prior to admission she was usually under alcohol's influence. The patient petitioned for her own one year's commitment, expressing a sincere wish to control her drinking. This sincerity lasted the three months of her hospitalization for she cooperated in all matters, and tended to be self-depreciatory in discussing her failures in life. She was classified in the group "Alcoholism without psychosis in a psychopathic personality." Four months after leaving the hospital she married a male fellow patient who also had been treated for alcoholism and both have managed exceedingly well for over five years.

Discussion

This study of fifty women addicted to alcohol indicates that certain personality traits tend to be prominent in spite of many different personality types within the group. Most of the women, prior to their alcoholism, were making average or better social adjustments and tended to be actively occupied. Most of them felt a vague dissatisfaction with their patterns of life, and the causes of this dissatisfaction tended to be varied and highly individual. Why the group turned to alcohol and continued with it, while others with similar problems do not, is not clear. Perhaps the problem must be attacked by studying people with similar problems and approximate personality traits in an effort to determine the factor that prevents the turn to alcohol.

The medical and psychiatric management of these patients within a mental hospital brought long term beneficial effects in about 20 per cent of the cases. European hospitals report cures of alcoholism up to 25 per cent. The American literature is almost totally free of such reports. It appears that only those patients who are fully cooperative in psychotherapeutic interviews stand to gain significantly by their hospitalization. In view of the addicts' marked and characteristic resistance to the psychiatric approach to their problems, methods of gaining their full cooperation must be devised. Even this may not prove sufficient, for the physiological effects brought about by the many years of drinking prior to hospitallzation may be a troublesome complication.

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Some Remarks on British Psychological Warfare*

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Each nation must take advantage of the psychological approach in this total war, but there are wide differences in the use of psychology made by Britain and Germany. The British application is less obvious and not as organized. The original use of psychology by the British grew out of the recognition of the German use of psychology and as a defense against it. Later the British developed their own methods and aggressive aims.

The chief difference between totalitarian and democratic propaganda is the difference between dictatorial agitation and liberal education. Both have propagandistic aims in common, that are based on the suggestibility of the masses. We have to thank Ernst Kris for the psychoanalytic interpretation of propaganda. He pointed out that it is based on suggestibility and thus is patterned according to the psychodynamic relations within the infantile situation. The child is dependent and exhibits the need for love and protection. These wishes may be dormant but never die and may be activated at any time with more or less ease. In a situation where dependence increases, suggestibility increases in proportion. As a rule the relations between the individual and reality are governed by the ego, but in the hypnotic spell or under the influence of suggestibility the function of the ego is taken over by the paternal image. In this situation the grownup person returns to the level of the child. Because of the desire for love and protection, orders and influence of the "leader" are accepted by the suggestible individual as if they resulted from his own ego. Obviously such identification cannot be a permanent one. It may change easily and unexpectedly with any alteration of the transferance situation between leader and follower. In this respect the identification based on suggestibility is quite different from that based on education. In the latter case, identification is more permanent because the ideas of the teacher are *incorporated* in and not only suggested to the ego.

Conditions of increased suggestibility are situations of ego weakness as for instance insecurity, anxiety, or conflicts which may arise when facing external dangers. Strengthening of the ego by acceptance and knowledge of reality will counteract suggestibility. Skepticism is a sign of a healthy and well functioning ego and as such a sure defense against the suggestions of propaganda.

It is apparent then how much easier it is for the individual in the totalitarian setting to become a victim of suggestions and propaganda than the sceptical individual in a free democratic sphere. The person in the totalitarian setting has lost his faith and accepts the new faith and the new dependence suggested to him. This is the end of the individual for from that point on he is told that he is without importance as an individual; that he is little and small; that he has to look for help to something super-personal—"the supremacy of the German race." This presents an analysis of Nazi propaganda. The German leader addresses followers, dependent and unthinking masses, in contrast to the democratic role where the leading person speaks in terms of "you and I" and not at a mass meeting, but from the fire-side, not suggesting but reasoning, not destroying but strengthening the individual, not aiming at the forming of a mass, but at the education

*Paper read at the Symposium of the Illinois Neuropsychiatric Institute, January 9, 1943.
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of the individual, not preaching a "new faith" but asking for a continuation of existing freedom.

The difference between the two methods is already discernible and becomes clearer as time goes on and the individual thinks out and digests the changing situations. The English were able to take defeat at Dunkirk. As a disappointed child loses faith in a mother who fails him, the German people will not be able to take Dunkirk or any serious setback. The transferance to the leader will disintegrate rapidly as it did in November, 1918. This is no secret to the German general staff and it was pointed out by none other than General Ludendorff.

Technical developments have helped immeasurably in formulating mass psychology and in creating suitable conditions for suggestibility. With the advent of radio it is possible to reach all individuals and to mold them into a gigantic mass, but this alone did not suffice. A total control of all means of propaganda is essential to create the most efficient use of suggestions. Propaganda is a double-edged sword; it is directed not only at the enemy, but also against its own people.

An outstanding example of English propaganda and methods of education is seen in the broadcasts of Edward Glover. They are collected in a book, "The Psychology of Fear and Courage." It is a small book and may be read over the week-end, but at the same time it is a great volume and contains many things to get enthusiastic about. The analyst talks to a nation and her allies. What he has to say and how he says it is an example of the part psychoanalysis has to contribute to the life struggle of the nation. Britain's "secret weapon" and her "secret armor" is her morale. To maintain it, two golden rules are given: First: Do not conceal from ourselves any real cause for uneasiness; and second: Detect and control those unreal alarms, suspicions and superstitions which tend to make us "weak-kneed and infirm of purpose during a crisis." These tenets are directly opposed to any premises on which suggestibility is based. It is not an appeal to the instincts to degenerate into submission, but it is an appeal for reality testing and as such an appeal for a strong ego and not for a weak suggestible one.

The British morale is a peace time morale and this, according to Glover, has to be changed. "We must become not only defenders but crusaders. Defense will consolidate a nation; only a crusade will carry it to victory. We must not be ashamed to develop for the time being a crusaders' passion." A fine broadcast given by Glover was "Women in Wartime." War conditions tend to drive a breach between the family and the state. This must be watched and controlled. Once the women are demoralized the end is near.

In the analysis of the problem "to hate or not to hate," Glover states: "We are fighting for our families, our lives, our cups and saucers, for our glass of beer, not to mention democracy. We are also fighting for the right to inward peace, for freedom from the blackout, from gas masks, from fear, and not primarily because we hate the enemy. We begin to hate the enemy when we realize that he threatens these possessions. From that point we begin to fight against something. But it would, perhaps, be truer to say that we are more indignant than hating. There is a kind of anger, slow to arouse, but which once aroused has of the energy of a boiling kettle." The war will be won *not* with propaganda but with education; not with seduction to regression but with an appeal to the reasoning power of free men; the war aim will not be the "supremacy of the super-man" but "realization of the American dream."

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Electro-Shock Therapy for Psychotic Epileptic

Case Report

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The recent application of electrically induced convulsions in the treatment of some types of mental disorders has been advocated and practiced by an increasing number of neuropsychiatric therapists. To date, however, no mention of the therapeutic use of electrically induced convulsions has been recorded in an epileptic patient. The following is an account of such electro-convulsive shock therapy used in treating a psychiatric reaction in an epileptic patient.

The use of electrically induced convulsions in the treatment of mental disorders was introduced by Cerletti and Bini.[1] Since their report the method has been used by several investigators in treating patients with schizophrenia, manic-depressive psychosis, involutional psychosis, depressive states and even chronic neurosis. Electro-shock treatment has been used in one or more of these conditions by Bogarello,[2] Groce and Cassiano,[3] Sogliana,[4] Kalinowsky,[5] Shepley and McGregor,[6] Fleming, Golla, and Grey,[7] Almansi and Impastato,[8] Gonda,[9] Myerson,[10] and many others. The number of favorable results has generally been proportional to the acuteness of the illness in contra-distinction to its chronicity. It has been possible more often to obtain remission and somewhat less often cure.

Case Report

Female, 22 years of age, convulsive seizures since the age of 3 years, psychotic behavior since the age of 18 years. Diagnosis: Epilepsy, idiopathic with psychosis (schizophrenic-like reaction).

E. L. V., a single girl with epilepsy was admitted to the hospital at the age of 20 years. The family history was neuropsychiatrically negative. There were no siblings. Patient was born one month prematurely, delivered by high forceps, weight 4 lbs., and was fed with a medicine dropper. There had been bilateral congenital cataracts which were needled at the age of 17 years with resultant visual improvement. The development and past history were non-contributory. Grand mal convulsions first occurred when the patient was 3 years old. During these the head turned to the right, the limbs became rigid, the patient would fall and convulse for a short time and then remain comatose for several minutes. Attacks recurred at intervals of 6 months at first, then at intervals varying from 3 to 4 weeks and on admission were occurring as often as twice weekly. Bromide therapy had caused a remission in attacks for three years while at home. The mother reported that at the age of 18 years the patient became excessively religious and a few months later exhibited odd behavior and expressed bizarre ideas. Soon thereafter she was confused a great deal of the time. She expressed hallucinatory phenomena stating that cinema actors were present in her home and talking to her, and therefore requested the family to keep quiet so that she could hear them. There were times when she refused fluid and food and would stand in one spot for hours. On one occasion she mistook the bathroom at home for a hospital and believed that there was a morgue in the cellar. She frequently called members of her family by a wrong name. On occasions she believed she saw extraneous objects in her food. Once when the family physician visited her bringing her some candy, she refused to eat it because she thought it was poison. On one occasion she accused her mother of cooking dogs to provide the family with food. She frequently ran through the house, searching the closets and removing all of the clothes onto the floor. Often she refused to remove her glasses and hat on retiring and would put on as many as six garments, one on top of another. Her bizarre phenomena were present irrespective of the occurrence of seizures.

On admission the patient exhibited essentially the same type of behavior as she had shortly prior to her removal from home. She was unresponsive, negativistic and did not respond to questioning. She refused to com-

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ply with simple requests unless guided by hand and with such guidance she would passively complete a movement. She imagined in succession that she was a tree, a dog or a Greek goddess. She exhibited cerea flexibilitas. There were occasional periods of overactivity lasting for several hours during which the patient sang snatches of songs, or spoke rapidly in an incoherent gibberish. It was often necessary to feed and dress her. There were frequent outbursts of aggression and destructiveness. She made unexpected and unprovoked attacks upon patients and hospital personnel, frequently refused to wear any clothing and because of her combativeness, kicking and biting, mechanical restraint, or seclusion, were oftentime found necessary. Her erratic behavior was unchanged by efforts at persuasion, psychotherapy, sedation and frequent family contacts. She received daily anticonvulsant therapy consisting of phenobarbital ½ grain and phenytoin-sodium 1½ grains three times daily. Her convulsize seizures averaged 5 grand mal and 1 petit mal attack monthly.

Physical examination revealed a girl of 5 ft. 4 in., weighing 107 lbs. Reflexes were hyperactive but equal. Bilateral corneal scars were present from previous needling for cataracts. There was a seborrheic dermatitis over the cheeks and sternum. Laboratory tests, including those of blood chemistry, urinalysis and spinal fluid examination were negative. A diagnosis of epilepsy, idiopathic with psychosis, was made.

Because of the schizophrenic-like behavior and its constant progression it was decided to administer electro-convulsive shock therapy. Maternal permission for this was readily given. A Reiter made machine was utilized. The morning of the treatment the usual anticonvulsant medication was omitted as was the breakfast. With commutator in operation, a current of sufficient milliamperage was gradually attained to produce a grand mal convulsion. During subsequent treatments a rise in convulsive threshold necessitated increasing the current to produce a convulsion. Gratifying improvement in behavior after a few shock treatments encouraged continuation of the treatment. The first day, a current of two milliamperes for two seconds did not provoke a convulsion and after a five-minute interval a current of three m. a. was applied for two seconds, a convulsion not ensuing until 15 minutes later. It was necessary to raise the milliamperage for subsequent treatment to the following milliamperage: 4, 10, 12, 16, 20, and 24 all maintained for two to three seconds. Improved behavior was noticeable after the fifth electro-convulsive treatment. The improvement was present from three to four days at a time following treatment. During this period there were none of the previous outbursts of aggression and destructiveness. The patient was more amenable to ward routine, took her meals willingly, did not remove her clothing nor make unprovoked attacks on other patients or hospital personnel. At times she reverted to her former unpredictable behavior but these episodes were admittedly milder and of shorter duration than before shock treatment. This improvement in behavior persisted for three months after the last shock treatment, but at this time was replaced by a renewal of negativism, aggression and nudity. At the request of the patient's mother and ward personnel, all of whom were impressed by the result of the treatment, a second series of electro-convulsive shock therapy was administered. The convulsive threshold was unaltered and as much current was now necessary as had been needed for the last treatment three months before, namely, 24 milliamperes for three seconds. The improved behavior which resulted the very day that the treatment was reinitiated convinced all concerned with the patient that shock treatment was the cause of the gratifying betterment. The outstanding feature of the patient's improvement was the abolition of aggressiveness and the institution of ready acquiescence to the ward routine. The second course of treatment comprised six shocks given on the basis of two shocks per week. Improvement persisted for 3½ months after the last shock treatment in the second series when a relapse occurred.

Although behavior was improved, neither the character nor the course of the usual spontaneous convulsions was appreciably affected by the electrically induced seizures. These spontaneous seizures occurred one to nine times monthly and, except for two instances of petit mal, were grand mal attacks. It was notable that status was never induced by electro-convulsive therapy, and that at all times only one grand mal convulsion resulted

from each application of electro-therapy. On a few occasions, one to two additional seizures occurred on the day of treatment.

Comment

In commenting upon this case there are several unusual features. Although electro-shock therapy has been applied recently on an extensive scale in psychiatric conditions, it had not been previously utilized for a psychiatric condition in a person with epilepsy. Whereas the spontaneously recurring epileptic convulsions did not ameliorate the psychotic reactions in this patient, the seizures electrically induced did favorably alter the psychotic reaction. Another unusual feature was the constant increase in the convulsive threshold with repeated electrically induced convulsions which contradicts the familiar and glibly repeated theory that one seizure predisposes to another. It was necessary to raise, not to lower, the milliamperage with succeeding treatment. The fact that the electro-convulsive shock therapy produced a single convulsion each time, rather than a series of seizures or status epilepticus, is likewise unusual in the light of the same theory that convulsions augment the convulsive state. These statements are facts, and while one case rarely suffices to promulgate a generality, it is evident that the oft accepted generality that seizures lower the convulsive threshold does not apply in this instance of electro-convulsive shock therapy administered to a patient with epilepsy and a schizophrenic-like psychosis.

Pacella and Barrera[11] compared the electro-convulsive thresholds in 6 epileptic patients with that in non-epileptic psychiatric patients, including those having paroxysmal cerebral dysrhythmia, and found the same electro-shock threshold in both epileptic and non-epileptic groups. But these authors reported that electrically induced convulsions did temporarily increase the number of spontaneous grand or petit mal epileptic seizures for a few days.

Summary

The use of electro-convulsive shock therapy in an epileptic patient with schizophrenic-like reaction ameliorated the psychiatric condition temporarily.

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Author's Note: Since this report was submitted, a second similar case has been treated, with improvement in behavior. In this case also, despite the epilepsy, there was no lowering of the convulsive threshold, no decrease in the effective convulsive milliamperage of 20, nor induced or spontaneous status epilepticus. The epilepsy was associated with tuberous sclerosis, and psychotic behavior.

Book Review

"PSYCHOMATIC MEDICINE (The Clinical Application of Psychopathology to General Medical Problems)" by Edwards Weiss, M.D., and O. Spurgeon English, M.D. Publishers, W. B. Saunders Co., Philadelphia & London, 1943. Ch. 23, 687 pages, including extensive references and index.

A readable book primarily designed for the general practitioner and internist rather than for the psychiatrically trained.

Personality development and general psychopathology are first considered, and then each physiological system is reviewed from the psychosomatic angle, using psychoanalytic concepts. The entire volume is liberally sprinkled with complete case histories demonstrating the inter-relationships between personality and somatic functioning. The importance of childhood experiences in terms of the de-

velopment of later emotional ills is well demonstrated. The psychosomatic aspects of "neuro circulatory asthenia," hypertension, digestive disturbances, the climateric, endocrine disorders, sexual disturbances, etc., are considered. Special topics, such as allergy, dental disorders of childhood, arthritis, are also reviewed from the psychosomatic point of view. There is one chapter devoted to the psychiatric aspects of military medicine. The last three chapters of the book are concerned with general principles of psychotherapy, treatment, and the training of those interested in psychosomatic medicine.

Although many of the psychoanalytic principles in this volume will confuse the general practitioner, he will beyond all doubt greatly profit by reading this well written treatise.

Kenneth Beach, M.D.
Galveston, Texas

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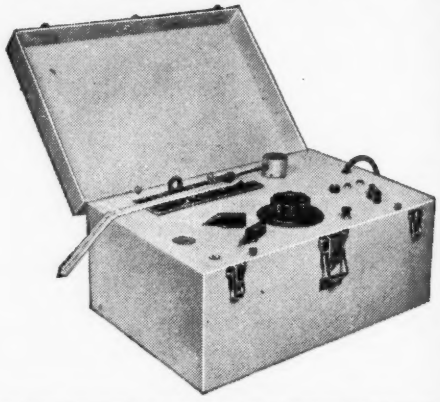

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